ACRP Advisory Committee on Radiological Protection

CCRP Comité consultatif de la radioprotection

Advisory Committees of the Atomic Energy Control Board of Canada — Secretariat: Ottawa, Canada K1P 5S9

Comités consultatifs de la Commission de contrôle de l'énergie atomique du Canada — Secrétariat: Ottawa, Canada K1P 5S9

COMMITTEE REPORTS

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From time to time the committees issue reports which are normally published by the AECB and catalogued within the AECB's public document system. Committee reports, bound with a distinctive cover, carry both a committee-designated reference number, e.g. ACRP-1, and an AECB reference number in the "INFO" series. The reports generally fall into two broad categories: (i) recommendations to the Board on a particular technical topic, and (ii) background studies. **Unless specifically stated otherwise, publication by the AECB of a committee report does not imply endorsement by the Board of the content, nor acceptance of any recommendations made therein.**

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Depuis les années cinquante, la Commission de contrôle de l'énergie atomique fait appel à des comités consultatifs composés d'experts indépendants pour l'aider dans ses prises de décision. En 1979, la CCEA a restructuré l'organisation de ces groupes de consultation pour former deux comités scientifiques supérieurs chargés de lui fournir des conseils indépendants concernant les principes, les normes et les méthodes générales touchant la radioprotection et la sûreté des installations nucléaires : ce sont le Comité consultatif de la radioprotection (CCRP), formé en 1979, et le Comité consultatif de la sûreté nucléaire (CCSN), établi l'année suivante. Le public peut consulter les comptes rendus des réunions de ces comités à la bibliothèque de la CCEA, à Ottawa.

Les comités rédigent à l'occasion des rapports qui sont normalement publiés par la CCEA et catalogués dans la collection des documents publics de la CCEA. Reliés avec une couverture distincte, les rapports des comités se reconnaissent à leur numéro de référence du comité d'origine (comme CCRP-1) et à leur numéro de référence de la CCEA dans la série «INFO». Ils se divisent habituellement en deux catégories générales : (i) les recommandations présentées à la Commission au sujet d'une question technique particulière; (ii) les études générales. **À moins d'indication contraire, la publication par la CCEA du rapport d'un comité ne signifie pas que la Commission approuve le contenu de la publication, ni qu'elle en accepte les recommandations.**

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ACRP Advisory Committee on Radiological Protection

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ACRP-12

Radiological Hazards to Uranium Miners

by the

Advisory Committee on
Radiological Protection

May 1990

An Advisory Committee of the Atomic Energy Control Board of Canada — Secretariat: Ottawa, Canada K1P 5S9

Comité consultatif de la Commission de contrôle de l'énergie atomique du Canada — Secrétariat: Ottawa, Canada K1P 5S9

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RADIOLOGICAL HAZARDS TO URANIUM MINERS

SUMMARY

The purpose of the present document is to review and assess the occupational hazards to uranium miners in Canada, with particular reference to amendments to regulations affecting the radiological protection of uranium miners (AECB 1986). These amendments set the maximum permissible dose to uranium minors at 50 mSv per year and state that the sum of radiation doses from all sources should not exceed this limit.

Uranium miners are exposed to radon and thoron progeny, external gamma radiation and long-lived alpha-emitting radionuclides in dust. There are many uncertainties in the estimation of lifetime risks from these exposures. The best value for the lifetime risk of inhaled radon progeny is about 3×10^{-4} lung cancers per WLM for the average miner, with a range of uncertainty from about $1 - 6 \times 10^{-4}$ per WLM. This central value is nearly twice as high as that recommended by the ICRP in 1981. The probability of serious biological consequences following exposure to external gamma rays is currently under review but is expected to be in the range of $3 - 6 \times 10^{-2}$ Sv^{-1} for workers. Dosimetric calculations indicate that the stochastic risks per WLM of thoron progeny are about one-third of those for radon progeny. The annual limits on intake of inhaled ore dusts that are recommended by the ICRP are probably too low by at least a factor of two for the type of ore and dust characteristics that are normally encountered in underground uranium mines in Ontario; this is due in part to the fact that the average diameter of these particular dusts is five times greater than the value used by the ICRP.

The magnitude of these different components in the sum of the radiological hazard varies greatly in different mines. Each component needs to be estimated for any given mining operation. Addition of the potential hazards from each individual component could result in a small overestimation of the risk since the risk estimates for radon progeny may include a small contribution from the other radiation sources. This conservatism appears acceptable from a regulatory viewpoint for protection of the health of workers.

Radiological exposures of uranium miners in Canada were reviewed. The biological impact of these exposures were compared with those of conventional accidents on the basis of time-loss detriment, that is, the years of normal life expectancy that are lost or seriously impaired due to occupational hazards. This comparison indicates that most of the time-loss detriment associated with uranium mining in Canada is due to conventional accidents rather than to radiological hazards. The objectives in considering all occupational risks are to reduce the total risk from all causes and to use funds spent for health protection as effectively as possible.

RADIOLOGICAL HAZARDS TO URANIUM MINERS

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RADIOLOGICAL HAZARDS TO URANIUM MINERS

1. INTRODUCTION

The 1977 recommendations of the International Commission on Radiological Protection state that the maximum permissible dose to atomic radiation workers should be 50 mSv per year and that the sum of the doses to all sources of radiation should not exceed this limit. Subsequent publications of the ICRP defined limits for inhalation of radon progeny by workers (ICRP 1981) and other concepts to be used in the radiation protection of workers in uranium mines (ICRP 1986). These recommendations are being incorporated into the new regulations which are currently being promulgated under the Atomic Energy Control Act of Canada (AECB 1986, 1989).

There has been considerable discussion of the validity of the particular limits recommended by the ICRP. The purpose of the present document is to review the available scientific information on the biological effects that might be anticipated in uranium miners in Canada under the new regulations. Uranium miners are exposed to a variety of radiation sources, notably radon progeny, thoron progeny, external gamma-rays, and dusts containing uranium, thorium and their progeny. All of these sources have to be considered. Other conventional hazards, such as traumatic accidents, silicosis, hearing loss due to high noise levels, development of "white fingers" (Raynaud's phenomenon) due to excessive vibration should also be considered when evaluating the total risk to workers in uranium mining and milling. An attempt should be made to keep all the risks as low as reasonably achievable and to make the best use of funds spent for safety, in order to obtain the maximum benefit for uranium miners.

The present document will review briefly the epidemiological data on risks of inhalation of radon and radon progeny by miners, the dosimetric and epidemiological data on the relative risks of exposure to other radiation sources, relevant data from animal experiments, current exposures of uranium miners in Canada, and compare the "conventional" and radiological risks to uranium miners with those to other workers in Canada.

S.I. units will be used throughout the document wherever possible, except in the case of exposure to radon progeny. The available epidemiological data on effects of exposure to radon progeny, as well as data on current exposures, are expressed in terms of working level months (WLM). 1 WLM equals exposure to a potential alpha energy from the short-lived daughters of 0.0035 $J.h.m^{-3}$. If the average breathing rate is taken to be 1.2 m^3 per hour, one WLM is equivalent to inhalation of short-lived radon progeny with a total potential alpha energy of 4.2 millijoules. For purposes of rough comparison, 1 WLM was previously taken to be approximately equal in risk to 10 mSv effective dose equivalent.

2. RADON AND RADON PROGENY

2.1 Epidemiological follow-up of miners exposed to radon progeny.

Epidemiological data on risks of fatal lung cancer in miners exposed to high concentrations of radon progeny in mine air were considered in a previous report (ACRP-1, 1982). The present review will concentrate on a number of important publications which have appeared since that time. These include the recent BEIR-IV report (1988).

The hazards of exposure of uranium miners to high concentrations of radon and radon progeny have been recognized for several decades. Since the radiation doses received after inhalation of radon alone are very small compared to doses from short-lived radon progeny, the health hazards are now attributed almost exclusively to inhalation of the progeny. Quantitative estimates of risk based on epidemiological follow-up of exposed miners have become increasingly available in recent years.

As in the case of the Hiroshima-Nagasaki bomb survivors, most of the exposed miners are still alive at present. In order to derive estimates of total risk of radiation-induced cancers over the complete lifetime of the exposed group of persons, some kind of risk projection models are required. Important factors to be considered for these models are the shape of the dose-response relationship, the latent period to appearance of excess cancers, effects of attained age and of time after exposure, and, in the case of lung cancer in particular, the confounding effects of cigarette smoking. These factors will be considered in the review which follows.

2.2 Risk coefficients from various epidemiological studies.

Risk coefficients are usually expressed in terms of "absolute risk", i.e. the excess number of lung cancers per 10^6 person.years at risk per WLM of exposure, or in terms of "relative risk", i.e. the percent increase in normal incidence of lung cancer per WLM of exposure. Table 1 summarizes some quantitative estimates of risk of induced fatal lung cancers per WLM of exposure to short-lived radon progeny. Lung cancers appearing less than 10 years after first exposure are usually excluded in these calculations in order to ensure better comparability of the data from short and long term follow-up and to ensure that the absolute risk coefficients are not too low because of the inclusion of years of follow-up during which little or no effect of exposure is anticipated (BEIR 1980). These data thus represent the average risk coefficients observed between 10 years after first exposure and the end of the follow-up, at which time most of the miners were still alive.

Other groups of miners demonstrating excess lung cancers as a result of exposure to high concentrations of radon progeny include the Joachimsthal-Schneeberg silver and pitchblende miners, French uranium miners, Cornish tin miners, Chinese tin miners, Swedish iron miners from Kiruna and Grangesberg, Swedish lead-zinc miners, Norwegian niobium miners (BEIR 1988) and Czechoslovakian iron miners and clay shale miners (Sevc 1988). In general, reliable estimates of risk coefficients cannot as yet be derived from these other studies.

A major problem in all the studies listed in Table 1 is the reliability of the exposure estimates. In general, exposure rates were highest during the earliest years of mining and were decreased, primarily by improved ventilation practices, in later years. However, monitoring of radon daughter concentrations was infrequent or absent during the earliest years of mining; the exposure histories of the early miners with the highest exposures and longest follow-up have necessarily been reconstructed from minimal data. In the study of 15,000 Ontario uranium miners, estimates of radon daughter concentrations were based on results of area monitoring in the mines in the early years or, in cases where no reliable measurements were available, on estimates provided by three mining engineers who were familiar with the Ontario uranium mines over the early years

of operation (Muller 1983). These estimates were derived by extrapolation from values measured at a later date, taking into account the ventilation rates, tonnage of ore produced and dust counts.

The risk coefficients derived from these studies (Table 1) vary over a range of about 10-fold. This is true both for estimates based on the relative risk model (% increase in normal incidence of lung cancer per WLM) and on the absolute or attributable risk model (excess number of lung cancers per 10^6 person-years at risk per WLM). Part of this variation is probably due to biases in estimated exposures.

The exposure estimates for the Swedish iron miners at Malmberget (Swent 1986, Chambers 1989) and for the Beaverlodge uranium miners (Chambers 1989, Frost 1989) are probably too low; the corresponding risk coefficients are therefore too high. The exposure estimates used by Howe (1986) in the study of Beaverlodge miners are too low because they are based on geometric rather than arithmetic means of measured concentrations of radon and radon progeny, and because they ignore additional exposures of Beaverlodge miners in other mines in this geographical area (Chambers 1989, Frost 1989). The exposure estimates used by Radford (1984) in the study of Malmberget miners are probably too low because they ignore changes in mining methods and changes from natural draft to mechanical ventilation during the years from 1930 to 1968 (Swent 1986, Chambers 1989). Most of the effort in assessment and re-assessment of exposure estimates has been devoted to the studies of the U.S., Ontario and Czech uranium miners and, more recently (Corkill 1984), of the Newfoundland fluorospar miners. These four studies could thus be considered to provide the most reliable risk coefficients at present. The risk coefficients derived from these four studies vary over a range of about 3-fold (Table 1).

2.3 Minimum latent period

Earlier analyses had in general not shown an appreciable excess of lung cancers within the first 10 years of follow-up after first exposure to radon progeny in mines (BEIR 1980). However, the studies of Beaverlodge and Port Radium miners both showed zero excess of lung cancers 0-5 years after first exposure but a statistically significant increase 5-10 years after first exposure (Howe 1986, 1987). Recent data on Czech uranium miners showed a statistically significant excess starting at the sixth year after first exposure (Sevc 1988). Analyses of epidemiological data on Ontario uranium miners also showed excess lung cancers in the period 5-10 years after exposure (Muller 1981,1989). The minimum latent period between exposure of miners to high concentrations of radon progeny and the first diagnosis of radiation-induced lung cancers is thus probably about 5 years (BEIR 1988).

2.4 Effect of time since exposure

A number of authors have attempted to analyze the epidemiological data on miners in terms of age at first exposure or of age at death (or diagnosis) from lung cancer. The effect of age at first exposure has been examined in 3 of the 4 Canadian studies. No consistent trends in the risk coefficients were observed (Howe 1986,1987, Morrison 1988). Three of the Canadian studies showed a trend towards a decrease in relative risk coefficient with increase in age at the time of appearance of lung cancers (Howe 1987, Morrison 1988, Muller 1985). A

similar trend is probably valid for the follow-up of U.S. uranium miners (Waxweiler 1981). This trend is very definite for the data on Czech uranium miners (Table 2).

In the general population, deaths from lung cancer are normally rare before age 40 and death rates tend to increase with a power function as the age at death increases from 40 to 75. If the relative risk model were more appropriate than the attributable risk model, one would expect to see a constant relative risk coefficient for younger and older age groups with a marked increase in absolute risk coefficient in going from the younger to the older age groups. The data from different studies are not consistent and thus do not provide good reason to reject one of these simple risk models in favour of the other.

The above models assume that the relative risk or absolute risk would remain constant throughout life after the minimum latent period. This assumption may be true for excess lung cancer incidence in the Hiroshima-Nagasaki survivors who were exposed to high doses of gamma-radiation in 1945 (BEIR 1988, UNSCEAR 1988). However, a number of publications now suggest that these simple models are not the most appropriate for uranium miners exposed to alpha-particles from radon progeny. A more sophisticated model was tested in a recent analysis of the Ontario data by Muller and co-workers (Muller 1989,1988). In this model, there is no increase in relative risk during the first 5 years after each increment in radon daughter exposure and best values for relative risk were calculated for subsequent times after each exposure. The available data on Ontario uranium miners suggested an increase in relative risk per WLM of 1.6% at 5-10 years, 3.4% at 10-15 years and 0.3% at 15 or more years after each annual increment in exposure (Muller 1988, 1989). For the purpose of calculating lifetime risk estimates, Muller (1989) assumed that the increase in relative risk would be zero at times greater than 15-20 years after each exposure to radon progeny. The number of radiation-induced lung cancers appearing at various times after exposure to 1 WLM per year for 5 years has been calculated using this model (Figure 1). The results suggest that exposure to 5 WLM between ages 50 and 55 might induce 100 times more lung cancers than would result from the same exposure between ages 20 and 25. The average lifetime risk of radiation-induced lung cancer for exposure to 1 WLM radon progeny per year from age 20 to age 55 would be close to 1.7×10^{-4} per WLM using this particular model and the particular risk coefficients derived in this study of Ontario miners (Muller 1989).

Other recent data have similarly suggested that risk varies appreciably with time since exposure. An analysis of U.S. uranium miner data indicated that the excess relative risk of lung cancer "fell dramatically in the years following cessation of exposure" (Hornung 1987). Data are also available for 4043 Czech uranium miners in study group S who started work in 1948-57 and terminated after an average of 10 years, i.e. about 1962 (Sevc 1988). Most of the excess lung cancers appeared in the period 10-27 years after first exposure (Sevc 1988) and apparently decreased to approach zero by about 33 years after first exposure, i.e. 23 years after the average termination of exposure to radon progeny in the uranium mines (Kunz 1988). More detailed analyses of the raw data have not yet been carried out, but the available data for Czech uranium miners (Sevc 1988, Kunz 1988) would appear to fit closely with the type of model suggested by Muller (1988,1989), though the risk coefficients would be higher for the Czech data than for the Ontario data.

Related analyses of epidemiological data on four cohorts, the Beaverlodge, Ontario and Colorado plateau uranium miners plus the Malmberget iron miners, were carried out by the BEIR-IV committee. As noted above, the exposure data for the Beaverlodge and Malmberget miners are not reliable. Moreover, the BEIR-IV analysis was based on time since last exposure in the mines, rather than on the preferable parameter of time after each annual exposure. However, the basic data are most valuable and are summarized in Table 3. This summary gives observed/expected (O/E) numbers of lung cancer deaths and % excess relative risk (E.R.R.) for all miners in each cohort excluding those listed with zero exposure. The excess relative risk decreases with time after last exposure for all 4 groups of miners, although not as drastically in the other groups as in the Ontario miners group.

The BEIR-IV committee analyzed the data for the four groups of miners in terms of age at first exposure, attained age at death, duration of exposure, and time since last exposure. Interpretation of the data in terms of possible interactions between these factors is obviously difficult. The final model chosen was a modified relative risk model in which the relative risk coefficient depends upon attained age (modifying factors were 1.2 for age below 55 years, 1.0 for age 55-64 and 0.5 for age 65 or more) and time since exposure. The increase in relative risk was assumed to be proportional to cumulative exposure in WLM incurred between 5 and 15 years before the attained age in question and to half the cumulative WLM incurred 15 years or more before this age.

2.5 Effect of cigarette smoking

Approximately 70% of the miners in all groups listed in Table 1 were cigarette smokers. The complete Ontario miners' study included large groups of gold miners and of copper-nickel miners as well as uranium miners. An excess of lung cancers was observed in the group of gold miners as well as the uranium miners, but not in the group of copper-nickel miners, who are believed to have a similar smoking history. The major cause of lung cancer among the gold miners was cigarette smoking, as expected, with a relative risk of 7 for smokers compared to non-smokers (Muller 1986). The excess lung cancers observed in the gold miners' group appeared to be correlated with exposure to high ore dust levels in the earlier years of mining before 1945. Smoking also increased the risk of silicosis (Muller 1986).

Interactions between cigarette smoking and inhalation of radon daughters were examined in the studies of Newfoundland fluorospar miners and of Ontario uranium miners. The data did not permit a clear choice between the relative risk model, indicative of a multiplicative relationship, and the absolute risk model, indicative of an additive relationship (Morrison 1988, Muller 1989). Other data have suggested that cigarette smoking accelerates the appearance of lung cancers in miners exposed to radon progeny, with the result that a multiplicative relationship between smoking and radon daughter exposure is most evident at the earlier stages of follow-up but may be absent or less evident many years after exposure (Hornung 1987, Archer 1979).

The epidemiological data on the Malmberget miners indicate that the risk of radiation-induced lung cancers in smokers was 1.4 times that in non-smokers (Radford 1984). The ratio of risks in smokers compared to non-smokers was 1.6 for the Czech shale-clay miners (Sevc 1988). A recent re-analysis of data on U.S. uranium miners suggests that the ratio of risks in smokers compared to non-

smokers was about 2 (Groer 1988, Chambers 1989). The expected ratios would be 1.0 for an additive model and 7 to 10 for a multiplicative risk model. Other data reviewed by the ICRP (1987) suggested the use of a relative risk model. However, as indicated in the 1980 BEIR-III report, it appears more probable that neither risk model is correct and that the true risk may be intermediate between those suggested by these two models. The 1988 BEIR-IV report used a modified relative or multiplicative risk model but indicated that a "submultiplicative model" may provide a more accurate description of the underlying relationship.

2.6 Dose-response relationship

The data from all 4 Canadian studies are compatible with a linear, non-threshold relationship between accumulated exposure and incidence of induced lung cancer. The fluorospar miners' study did not provide any evidence of a decreased response per unit dose at high accumulated exposures in the region of 2500 WLM (Morrison 1988), such as has been observed in the study of U.S. uranium miners (BEIR 1980, Hornung 1987, Archer 1979). A recent report suggests that the apparent curvature in the reported dose-response relationship for the U.S. uranium miners may be an artefact due to a systematic bias in the measurements of radon progeny during the years 1951-1953 (Schiager 1989), and that the two groups of U.S. miners with the highest exposures should not be used for making risk estimates. This conclusion would remove much of the evidence for curvilinearity in the dose-response relationship previously reported for the U.S. miners. The overall Czech data for group S are also compatible with a linear dose response relationship, although there is some suggestion of decreased response per unit dose at cumulative exposures above 300 WLM (Sevc 1988).

2.7 Lifetime risk estimates for fatal lung cancers

In most cases, estimates of the lifetime risk of radiation-induced lung cancer are necessarily based on extrapolation from epidemiological observations over a limited period of time to projections over the total lifetime of the irradiated cohort, most of whom are fortunately still alive at the time of observation. The particular risk projection models chosen are important for this reason. Four previous estimates of lifetime risk as derived from epidemiological studies on adult male miners are given below:

ICRP Publication 32 (1981)	$1.5\text{-}4.5 \times 10^{-4}$ WLM^{-1}
ACRP-1 (1982)	$1\text{-}6 \times 10^{-4}$ WLM^{-1}
BEIR-IV (1988)	5×10^{-4} WLM^{-1} [males exposed from birth]
UNSCEAR (1988)	$1.5 - 4.5 \times 10^{-4}$ WLM^{-1}

The first two and the fourth estimates suggest an average risk of about 3×10^{-4} WLM^{-1} for uranium miners. The BEIR-IV estimate is for males exposed from birth but would be similar for adult males exposed from age 20 to 60. According to Cross (1988), the BEIR-IV analysis suggests a risk of 1.4×10^{-4} WLM^{-1} for adult human male non-smokers. The BEIR-III report did not provide an estimate of lifetime risks.

The epidemiological data on uranium miners were also reviewed in ICRP Publication 50 (1987). The most appropriate average risk coefficients were considered to be about a 1% increase per WLM in normal lung cancer incidence (relative risk

model) or about 10 excess lung cancers per 10^6 person-years at risk per WLM (absolute risk model). These values are compatible with the data on Ontario uranium miners but are rather low for the data on Czech uranium miners (Table 1). In two recent Canadian studies, the excess risk of death from lung cancers before age 70 has been calculated for male uranium miners using a life table approach, where the expected values for non-exposed males were derived from Canadian vital statistics (Table 4).

The calculated values thus apply to a population whose survival is comparable to that of persons of the same age and sex in the general population of Canada. Using the average risk coefficients suggested in ICRP Publication 50 (1987), the values are equivalent to a risk of 2.5-2.9 x 10^{-4} per WLM to age 70. A total lifetime risk of about 3 x 10^{-4} per WLM would appear to be a reasonable choice, assuming that exposure of adult male miners to radon progeny at any age resulted in an increased risk of lung cancer which continued throughout life. If the relative risk model were correct, the risk to non-smoking miners would of course be appreciably smaller (ICRP 1987, Saccomanno 1986, BEIR 1988).

Assuming that increased risk ceased 15-20 years after each exposure, the lifetime risk would be closer to 2 x 10^{-4} per WLM for male miners exposed to 1 WLM per year from age 20 to age 60, based on the time-dependent relative risk coefficients which were derived from the study of Ontario miners (Muller 1989). Further study of other groups of miners and of miners exposed only at younger ages would be required to substantiate the risk coefficients for this latter model. Exposure of miners at a constant rate for 40 years is of course unlikely to occur in uranium mining, but some assumption of this kind is required in order to derive average lifetime risk estimates.

Approximate lifetime risk estimates for group S of the Czech uranium miners can also be derived directly from the data without the use of any risk projection models. Follow-up data to the end of 1980, when excess lung cancers were apparently declining but were still appreciably above zero, indicate 386.5 excess lung cancers in 4043 miners exposed on average to 226 WLM (Sevc 1988). This represents a risk of 4.2 x 10^{-4} WLM^{-1} at 18 years after the average last exposure. The data available to date for another 5 years of follow-up (Kunz 1988) suggest that the extrapolated lifetime risk will approach 5 x 10^{-4} WLM^{-1} for group S of the Czech uranium miners. This value is similar to the projected lifetime risk of 4.5 x 10^{-4} WLM^{-1} suggested for the Czech uranium miners in the UNSCEAR report of 1977 and in ICRP Publication 32 (1981).

The analyses of epidemiological data available to 1988 do not suggest any reason for a major change in the range 1-6 x 10^{-4} WLM^{-1} (ACRP 1982), or in the average value of about 3 x 10^{-4} WLM^{-1}, for the lifetime risk of fatal lung cancer following exposure of adult male miners to high concentrations of radon progeny in the air in uranium mines. This average value, based on epidemiological data only, is of course nearly twice that suggested earlier by the ICRP (1981) and the NCRP (1984) after consideration of both epidemiological and dosimetric approaches.

2.8 Other hazards of exposure to radon progeny

There is no reproducible epidemiological evidence of excess fatal cancers other than lung cancers among miners who were previously exposed to high concentra-

tions of radon progeny. Calculations of doses to the gonads and other tissues after inhalation of radon progeny also indicate that these doses should be very small by comparison with doses to the respiratory tract (ICRP 1981, Myers 1985, Jacobi 1980, NEA 1983). The risk of induction of lung cancers is therefore considered to represent essentially all of the risk of serious health hazards attributable to inhalation of short-lived radon daughters.

An increased incidence of curable skin cancers (basal cell carcinomas) has been reported for the Czech uranium miners (Sevc 1988, Sevcova 1978). No comparable increase was observed for surface workers at the uranium mines or for underground coal miners or surface workers at the coal mines in Czechoslovakia. The increased incidence among the uranium miners was attributed to contamination of the skin surface by radon progeny. The approximate risk estimate given (Sevc 1988) is compatible with recent estimates suggested in ICRP Publication 45 (1985). Data on incidence of curable skin cancer among uranium miners in other countries are not available for comparison; the Czech data are unique in this respect.

2.9 Other radiation sources as a confounding factor

Uranium miners are exposed to a variety of radiation sources in addition to inhaled radon progeny notably, external gamma-rays from the radioactive ore, inhaled thoron progeny and inhaled uranium and thorium ore dusts. It is unlikely that these other sources made a major contribution to the excess lung cancers produced in uranium miners at a time when the concentration of radon progeny in the mine air was very high. The external gamma ray exposures depend on the concentration of radioactive material in the ore body and would remain unchanged by the increased ventilation which has typically reduced the concentration of radon progeny in mine air by factors of 10 to 100-fold since the 1950's. Thoron progeny represent an appreciable radiological problem in some underground mines, notably at Elliot Lake in Ontario; however, their concentration is affected less than the concentration of radon progeny by increased ventilation (Johnson 1985). The increased ventilation rates and other practices which have been introduced since the 1950's may have had less effect upon ore dust concentrations than upon the concentration of radon progeny in the mine air (Ham 1976). For example, dust levels in Ontario uranium mines decreased 1.8-fold between 1960 and 1975 (Ham 1976) while the estimated concentrations of radon progeny decreased 5-fold over the same period of time (Muller 1989). Other data suggest that the levels of ore dust and of radon progeny in the mine air decreased to similar extents over the years in the Ontario mines (Table 5).

After consideration of these confounding factors, the ICRP (1987) indicated that "the observed excess lung cancer frequency among the study groups of uranium miners is mainly attributable to the inhalation of short-lived ^{222}Rn daughters. The risk contribution from inhaled long-lived radionuclides and gamma-radiation might be, on the average, about 10-20% of the total" (ICRP 1987). This conclusion appears reasonable. However, it should be noted that the radon progeny in the air in the Newfoundland fluorospar mines and in the Malmberget iron mines arose not from the ore itself but from dissolved radon carried into the mines by seepage water; thus the excess lung cancers observed in these two groups of miners (Table 1) cannot be attributed to radiation sources other than radon progeny.

Because of other major uncertainties in the derivation of lifetime risk estimates for inhaled progeny (see sections 2.1 to 2.7 above), the correction for other radiation sources as a confounding factor would appear to play a relatively minor role. It should nevertheless be noted that if a single value of about 3×10^{-4} fatal lung cancers per WLM were selected as the best lifetime risk estimate derived from epidemiological follow-up of miners, the true value for radon progeny alone would be closer to $2.4\text{-}2.7 \times 10^{-4}$ per WLM (ICRP 1987).

Exposure of miners to radon progeny in homes and other locations outside the mine is not taken into account in derivation of the risk estimates given above. It is unlikely that average exposure of miners from non-occupational sources would have exceeded 1 WLM per year (as compared to average values of about 0.1 to 0.15 WLM per year for the general population). Occupational exposures to 20 to 50 WLM per year were not uncommon in the early years of uranium mining in Czechoslovakia, Ontario, Newfoundland and the USA. The errors due to neglect of radon progeny from non-occupational sources are likely to be small, at least for these particular study groups. This topic is being explored in Czechoslovakia (Chambers 1989).

The February 1990 draft of proposed new ICRP recommendations does not review hazards of radon progeny in great detail but suggests that the probability of fatal lung cancers would be about $1\text{-}4 \times 10^{-4}$ WLM^{-1}, or an average of about 2.5×10^{-4} WLM^{-1}.

3. OTHER RADIATION SOURCES

3.1 External Gamma Radiation

Epidemiological data on the health effects of X- and gamma-rays have been reviewed many times by scientific committees (BEIR 1980,1990, ICRP 1977, UNSCEAR 1977,1982,1986,1988). For purposes of radiation protection, it has in the past generally been assumed that exposure of radiation workers to 10 mSv of whole body radiation is associated with an average lifetime risk of about 1.3×10^{-4} fatal cancers in the workers plus a risk of 0.4×10^{-4} serious genetic diseases in the children and grandchildren of the exposed workers (ICRP 1977). These values are near the middle of the ranges of the risk estimates suggested by the 1980 U.S. BEIR committee. The suggested values are currently being reviewed by various committees in view of recent changes in estimates of absorbed doses received by atomic bomb survivors at Hiroshima and Nagasaki in 1945 (ICRP 1987). Final reports on this topic are not anticipated for some time. Risk estimates for external gamma radiation are also being reviewed by a separate working group of the ACRP.

The 1988 UNSCEAR report derived a lifetime risk estimate of 4-8 fatal cancers for a working population of 100 persons exposed to 1 Gy low LET radiation at high dose rate. It was suggested that this value should be reduced by a factor of 2 to 10 for low doses at low dose rate. This reduction would yield a risk estimate of 0.4-4 fatal cancers per 100 person-Sv. The 1990 BEIR-V report gave a lifetime risk estimate of 6 fatal cancers per 100 person-Gy for workers and suggested possible reductions by 2 to 5 times for fatal cancers other than leukemia. This reduction would yield a risk estimate of 1.9-3.5 fatal cancers per 100 person-Sv to workers. The above values can be compared with the risk estimate of 1.25 fatal cancers per 100 person-Sv given by the ICRP in 1977. There are, of course, appreciable uncertainties in all of these estimates. The

possibility that low radiation doses might induce zero fatal cancers cannot be excluded (AMA 1989, Gilbert 1989, BEIR 1990), even though this possibility is usually ignored for radiation protection purposes. An estimate of 4×10^{-2} fatal cancers per person-Sv to workers would represent a reasonably conservative value that could be derived from the recent reviews in the UNSCEAR (1988) and BEIR (1990) reports. This value is the same as that given in the February 1990 draft of proposed new ICRP recommendations.

Whole body radiation is also expected to cause an appreciable number of non-fatal or curable cancers, primarily cancers of the skin, thyroid gland and female breast (BEIR 1980, ICRP 1977, UNSCEAR 1977). These curable cancers were not considered in the derivation of weighting factors for various tissues by the ICRP in 1977. Curable cancers could on average be assigned roughly one-eighth the weight of fatal cancers (ICRP 1985). The total weighted risk of serious health effects resulting from exposure of radiation workers might then be taken to be about 6×10^{-2} per Sv (4×10^{-2} per Sv for fatal cancers only) after allowing for future increases in risk estimates as a result of changes in the Hiroshima-Nagasaki data, changes in models used to estimate lifetime risks, and inclusion of genetic diseases in all future generations rather than the first two generations only. The potential health hazard associated with a whole body exposure of 10 mSv from gamma radiation is thus expected to be somewhat larger than that associated with an exposure of 1 WLM of radon daughters. These estimates apply to adult populations only. Complications and uncertainties associated with exposures of populations that include young children are not considered in this report.

3.2 ALIs for Thoron Progeny and Radioactive Ore Dusts

No epidemiological data exist for direct estimation of the risk of exposure to thoron daughters or to radioactive ore dusts. Potential health hazards from thoron progeny and radioactive ore dusts can only be estimated by using the results of dosimetric calculations to relate risks from exposure to these substances to risks from exposure to radon progeny or from exposure to X- or gamma-rays. For intakes of radionuclides, exposures are related to a common quantity, the committed effective dose equivalent (CEDE) or the annual limit on intake (ALI) (ICRP 1977). The limits recommended by the ICRP are summarized in Table 6. These limits were intended to result in a CEDE of 50 mSv to Reference Man. A lower ALI for thorium ore dust, based on the concept of limiting the committed dose to bone surfaces to 500 mSv, is recommended by ICRP (1986). This limit is meant to restrict doses to values below those that might result in non-stochastic effects. Since this report is primarily concerned with the induction of cancer and genetic effects (i.e. stochastic effects), the ALIs based on the dose limit for stochastic effects are given in Table 6.

There are some important differences in the calculations of effective dose equivalents from which the annual limits on intake are derived. Dosimetric calculations for inhaled radon progeny which are based on a weighting factor of 0.12 for the whole lung (ICRP 1977) suggest a CEDE of about 4 mSv per WLM (ICRP 1981), i.e. a lifetime risk of about 0.7×10^{-4} lung cancers per WLM. This is appreciably lower than the lifetime risk of $1.5\text{-}4.5 \times 10^{-4}$ per WLM as estimated from epidemiological studies (ICRP 1981). Calculations of CEDEs for radon and thoron progeny were therefore based on the regional lung concept (ICRP 1981, Jacobi 1980, NEA 1983), in which the tracheo-bronchial and pulmonary regions of the lung (Fig. 2) were each assigned a weighting factor of 0.06 compared to the

weighting factor of 1.0 for whole body irradiation. The average CEDE for inhaled radon progeny in the mine environment would then become about 8 mSv per WLM (NEA 1983). Using these weighting factors, most (nearly 90%) of the health hazards of inhalation of radon progeny appears to be associated with irradiation of the tracheo-bronchial region (NEA 1983). Most of the potential hazard of cancer due to inhalation of thoron progeny would also appear to be due to irradiation of the tracheo-bronchial region; however, the relative long half-life (10.6 hours) of ^{212}Pb (thorium B) allows appreciable time for translocation of this daughter to other tissues in the body. Thus about 30% to 40% of the total hazard (or, more precisely, total CEDE) associated with inhalation of thoron progeny is thought to be due to irradiation of tissues other than the lung, notably bone surfaces and kidneys (NEA 1983).

The ALIs for ore dust, on the other hand, were based on the mean lung concept, in which the whole lung is assigned a weighting factor of 0.12 (ICRP 1977). Suggested refinements (Johnson 1985) of the CEDE calculations, including application of the regional lung concept, would lead to ALIs which are appreciably higher than those given in Table 6.

Some of the uncertainties involved in the derivation of ALIs have been considered in the NEA Experts Report on dosimetry aspects of exposure to radon and thoron progeny (NEA 1983) while other problems are currently being considered by the ICRP Task Group on respiratory tract models (Bair 1987). The major uncertainties are reviewed below.

3.3 Deposition in the Lungs

The main factors affecting deposition of inhaled radon and thoron progeny in lungs are breathing rate, unattached fraction of daughters, and size (plus geometric standard deviation of size) of the aerosol particles for the attached fraction of radon and thoron daughters. The effective dose equivalent will vary about a factor of two over the range of these parameters that can reasonably be expected to represent the long-term average in underground mines (NEA 1983).

In the case of inhaled ore dusts, ALIs calculated with the current lung model vary over a 7-fold range as the average particle size changes from 0.5 μm to 10 μm (Johnson 1985). The ICRP recommendations (1986) are most appropriate for 1 μm particle sizes and probably overestimate the presumed toxicity of ore dusts with larger particle sizes, since these tend to deposit in the naso-pharyngeal region (Johnson 1985,1987). Average particle sizes for ore dusts in the Elliot Lake mines in Canada appear to be about 5 μm, which would increase the ALIs in Table 5 by a factor of about 2.5 (Johnson 1985). Revisions to the current lung model by the ICRP Task Group (Bair 1987) suggest that even at 1 μm diameter, deposition is 2-fold lower than previously recommended (Johnson 1987), i.e., the ALI is too low by a factor of 2.

3.4 Retention of Deposited Material

The main factor governing retention of radon progeny, and to a lesser extent thoron progeny, is radioactive decay. Since the total transit time of mucus through the tracheo-bronchial region of the lung (Fig. 2) is between 0.25 and 2 days, most of the radon progeny will decay in the tracheo-bronchial region, whereas a considerable fraction of the thoron progeny could leave the tracheo-bronchial region before decay. This range in retention could result in a

variation in CEDE of less than two-fold for radon progeny, and of four-fold or greater for thoron progeny (NEA 1983). There is also a possibility that some of the activity may be transported into the epithelium of the tracheo-bronchial region. Although the dosimetry used by ICRP (1981) has not considered this possible increase in dose, the additional contribution to the average dose to target cells in the respiratory tract is probably not very large.

Because of the long half-lives of the alpha-emitters in uranium and thorium ore dusts, a major fraction of the accumulated radiation dose to the lungs from inhaled dusts is delivered to the pulmonary region (Fig. 2) rather than the tracheo-bronchial region. Other tissues which receive appreciable radiation doses after inhalation of ore dusts include liver, kidney, bone surfaces and red bone marrow (Johnson 1985). Previous calculations of doses received after inhalation of ore dusts have assumed that the alpha-emitters in the ore dusts were highly insoluble class Y materials (Johnson 1985). Recent studies indicate that uranium ore dust in Canadian mines may be more soluble than Class Y (Johnson 1987, Robertson 1986, 1988). Measurements of the solubility of various ore dusts in simulated lung fluid indicated that about 40% of the uranium and a lesser amount of the other radionuclides were removed by dissolution of the ore dust within two weeks. Measurements of the translocation of the radionuclides in ore dust from the lung to other organs in a study of instilled ore dust in rat lungs (Hicks 1988) have confirmed the in vitro results. If ore dusts are more soluble than has been assumed, the dose to lungs will be lower, and those to other organs will be higher, than previously calculated. However, this will have only a minor impact on the ALI as the ALI is based on the weighted sum of doses to all organs, and the decrease in lung dose is approximately offset by an increase in doses to other organs.

3.5 Calculation of Absorbed Dose

The initial calculation of absorbed dose from the integrated activity of deposited radon and thoron progeny in the tracheo-bronchial region was done by ICRP (1981) by assuming that cells at risk were distributed in the epithelium at a depth below the cilia ranging from 30 to 60 μm at the upper air-ways to 10 to 20 μm in the last bronchiole generation. There is considerable uncertainty in these values, and hence in the dose, particularly in the upper airways. It is noted that the absorbed dose to the tracheo-bronchial cells calculated by ICRP is the average over all generations of the tracheo-bronchial tree; this average is less variable than is the calculated dose in any given airway generation. This is a major difference between the ICRP (1981) and the U.S. NCRP (1984) dosimetry as the NCRP only uses the dose to the cells at a depth of 22 μm in the fourth airway generation. This makes the ICRP calculations less sensitive to variations in unattached fraction, to the particle size distribution of the attached fraction, and to uncertainties in cell depth than the NCRP calculations. Revisions of the lung model currently being considered by the ICRP Task Group (Bair 1987) include averaging the dose over all of the bronchial epithelium over all generations in the tracheo-bronchial region. This revision would not cause any major change from that currently calculated (ICRP 1981) in the mean absorbed dose to the tracheo-bronchial region.

In the case of ore dusts, the absorbed dose to the lung was based on the average dose from activity in the tracheo-bronchial and pulmonary region and the associated lymph nodes. However, activity tends to concentrate and be retained in the lymph nodes, which are less radiosensitive than many other tissues in the

body; the net effect of corrections for this factor would be a decrease in potential risk (Johnson 1985). In those calculations, Johnson (1985) assumed that retention in the tracheo-bronchial region, and hence dose to this region, was small. However, more recent data point to the possibility of significant retention in the tracheo-bronchial region, and doses to this region may be comparable with those to the pulmonary region. Calculations of absorbed doses from inhaled activity which is translocated to the bone also requires further consideration of the distribution of this activity in the bone. The assumed distribution of uranium and radium in bone is likely realistic but thorium is assumed to be deposited on bone surfaces, and to remain there indefinitely. There has been no detailed studies of the thorium distribution in bone but if it acts similar to other actinides, it will become buried in mineral bone during bone remodeling, and the dose to bone surfaces and red bone marrow will be significantly reduced (ICRP 1986). Priest and Birchall (1985) have calculated that this remodeling results in a decrease in the bone surface dose by a factor of 6 and in the red bone marrow dose by a factor of 3 for plutonium. Similar reductions could be expected for thorium (Johnson 1985). Decreases in the doses of this size will result in an increase in the ALIs given in Table 6 by about a factor of 2 for thorium ore dusts, and a smaller increase for uranium ore dust.

3.6 Conversion of Absorbed Dose to EDE

The conversion of absorbed dose to dose equivalent and thence to EDE requires the selection of a quality factor (Q) and weighting factor (W_T). The quality factor based on the strict definition of radiation quality used by the International Commission on Radiation Units and Measurements (ICRU) is greater than 20; however, ICRP (1977) uses 20 as the limit on Q because very high quality (high LET) radiation is more likely to result in cell death than in a viable but damaged cell. Using similar arguments, Hoffman and Katz (1983) derived a quality factor of less than 10 for alphas, and work at the U.K. National Radiological Protection Board on chromosome aberrations in lymphocytes would tend to support this value (Edwards 1985). However, data on induction of lung cancer in animals by radionuclides deposited in the lung suggest that alpha emitters are about 30 times (range 6 to 40 times) more effective than beta-gamma emitters (ICRP Publication 31, 1980). A Joint Task Group of the ICRP and ICRU has recently recommended a Q of 25 for alpha-particles (ICRU 1986).

The weighting factor of 0.12 assigned to the whole lung is derived from epidemiological data (ICRP 1977). As with all epidemiological data (see for example Table 1), there are uncertainties associated with this number; these uncertainties can only be minimized by further follow-up of exposed populations. The regional lung concept, in which half the weighting factor for the whole lung was assigned to the tracheo-bronchial region and half to the pulmonary region, was introduced in order to arrive at some reasonable dosimetric estimates of the potential toxicity of inhaled radon and thoron progeny (ICRP 1981). However, "human cancers induced by cigarette smoking or exposure to environmental agents nearly always arise from epithelium in proximal regions of the bronchial tree" (BEIR 1980). It has been suggested therefore that about 80% of the weighting factor for the whole lung should be assigned to the tracheo-bronchial region and only 20% to the pulmonary region (Masse 1987). This change would result in an increase of up to 70% in calculated CEDE's for radon and thoron progeny; the average CEDE for inhaled radon daughters would then be closer to 14 mSv per WLM than to the previously suggested value (NEA 1983) of 8 mSv per WLM. The potential hazard from inhaled radon progeny as derived from dosimetric calcula-

tions would, under these circumstances, approach more closely to that derived more directly from epidemiological studies. The suggested change in weighting factors could, on the other hand, result in a decrease in calculated CEDEs for inhaled ore dusts retained in the lung. The amount of this decrease will be entirely dependent upon the amount of retention of these ore dusts in the tracheo-bronchial region of the lung. The appropriate value to use is currently under discussion by the ICRP Task Group on Lung Models (Johnson, personal communication). However, any significant decrease in calculated CEDEs for inhaled ore dusts could increase the ALIs for very insoluble uranium and thorium ore dusts by about 5-fold. The increase would be less for more soluble ore dusts because of contributions to the CEDE from radionuclides transferred from the lung to other tissues in the body.

It might be noted that the dosimetric calculations provide a strong link between the potential toxicities of radon and thoron progeny, which is independent of any uncertainties in the best values for Q or for weighting factors. The ratio of the risks of lung cancer induced by radon progeny and by thoron progeny does not depend upon the particular values of Q and W_T that are selected.

4. STUDIES ON ANIMALS

Human epidemiological data provide the most important basis for assessing the risk from exposure to radon and to external gamma radiation. However, additional insight into the nature of the exposure-response relationship can be obtained from animal experiments. The past exposures of underground miners to radon progeny are uncertain, and animals can serve as surrogates in studying various aspects of the radon exposure problem.

4.1 Animal Studies With Gamma Rays

Many animal studies on induction of cancer and genetic changes by external gamma-radiation have been carried out. These studies have been thoroughly discussed in UNSCEAR reports from 1972 to 1988 and will not be reviewed in detail in the present document. The primary conclusions that might be drawn from these studies include the following: (a) Many different types of cancer can be induced in diverse animal tissues by irradiation. (b) The sensitivity to induction of a given type of cancer varies appreciably from one species to another and even from one strain of a given species to another. (c) Lifespan shortening at low doses and low dose-rates is due to cancer induction; a wide variety of other non-specific effects may contribute to lifespan shortening at high doses above 3 Sv. (d) Gamma radiation is 2 to 5 times less effective at low dose rate than at high dose rate for induction of cancer (NCRP 1980, UNSCEAR 1988, BEIR 1990); this conclusion does not apply to high LET radiations such as alpha-particles. The magnitude of this dose rate effectiveness factor is critical to assessment of risk estimates for external gamma rays (see section 3.1) but will not be considered in detail in this document.

4.2 Animal Studies with Radon Progeny

Several biological effects have been observed in dogs and rodents following the inhalation of radon and radon progeny at exposures above 5000 WLM: respiratory epithelial carcinoma, pulmonary fibrosis, pulmonary emphysema and lifespan-shortening. Lesions above the trachea (extrapulmonary lesions) and nonpulmonary lesions have been reported in these studies, but their significance with respect

to human exposures below 600 WLM is unclear. No significant lifespan shortening or pulmonary fibrosis was observed in rats with cumulative exposures below 600 WLM (Cross 1988).

Five variables appear to influence the tumorigenic potential of radon progeny in the experimental animal studies:

- cumulative exposure
- exposure rate
- unattached fraction
- disequilibrium between radon and its progeny
- concomitant exposures to cigarette smoke.

The tumorigenic potential of exposure to radon progeny increases with: a) increase in exposure, and b) increase in unattached fraction and disequilibrium. Although simultaneous exposure to elevated levels of ore dust or diesel-engine exhaust increased the incidence of emphysema and fibrosis, it did not appear to increase the number of tumors produced by radon decay products (Cross 1988). Thus, radon decay products alone are able to produce lung carcinomas. The data from the Pacific Northwest Laboratory suggest that the risk of lung cancer in rats is not greatly influenced by dose rate between 5 WLM and 500 WLM per week for cumulative doses below 2000 WLM (Cross 1988) (Figure 3).

The influence of cigarette smoke depends on the temporal sequence of the exposures. In beagle dogs, alternating exposure to cigarette smoke and radon progeny on the same day produced a decrease in lung-tumor incidence from that produced by radon progeny alone. Exposure to smoke before exposure to radon progeny did not alter the lung-cancer incidence in rats; however, exposure to smoke following exposure to radon progeny produced a synergistic effect in studies originally carried out in France (Chameaud, 1976, 1981) and which were later confirmed by Cross (1988). This is the type of result that would be expected if cigarette smoke acted primarily as a promoting agent, as it does for radiation-induced epithelial tumors in rat skin (McGregor 1982). The influence of exposure to other pollutants is unknown at the low levels of radon progeny currently present in the mines and in the environment.

Although lung-tumor data differ somewhat, the overall incidence data are similar for adult male rats and man. The derived range in mean lifetime risk coefficient for exposures of rats to 100 to 5000 WLM at weekly rates exceeding 5 WLM, with low unattached fractions and with data uncorrected for lifespan differences from control animals, is about 1 to 5 x 10^{-4} WLM^{-1} (Cross 1988). Similar cumulative exposure data from underground miners are also estimated to yield risk coefficients between about 1 and 5 x 10^{-4} WLM^{-1}. Other recent data from rats, with exposures of 20 to 50 WLM at low exposure rates, place the lifetime risk coefficient at about 6 to 8 x 10^{-4} WLM^{-1} (Cross 1988). It should be noted that the risk coefficients for rats include all lung tumors, both benign and malignant (Cross 1988), while those for humans include malignant tumors (cancers) only. With this proviso, estimated lung-tumor risks per WLM are about the same in humans and in rats. Hamsters, on the other hand, are relatively resistant to the carcinogenic action of inhaled radon progeny (Cross 1988).

Observations in animals that have not been reported in humans are: a) the increase in tumor incidence with increase in unattached fraction and disequilibrium, b) the importance of the temporal sequence of exposure to cigarette smoke

and radon progeny, and c) the presence of extrapulmonary lesions, including carcinoma. (The absence of these findings does not constitute proof that similar data might not be obtainable in humans but may rather reflect the paucity of this type of human data (Cross 1988).)

The similarities in the human and animal data presently outweigh the differences between them. The animal experiments continue to yield valuable information that helps to explain the effects on man of inhaled radon progeny and associated pollutants, especially in mine environments.

4.3 Animal Studies With Other Alpha Emitters

Besides uranium miners and other miners who were exposed to high concentrations of radon progeny in the past, there are no human populations that have shown health effects that can be attributed to inhalation of radionuclides (ICRP 1980). Thus animal experiments provide virtually all of the information about the biological effects of inhaled radionuclides other than radon progeny.

Studies with inhaled uranium ore dusts in rats are currently being carried out at the Battelle Pacific Northwest Laboratory (Cross 1988) and other studies are being funded by AECB in Canada. Since these studies are incomplete, it is not yet possible to evaluate the results.

Extensive studies have been carried out in rats, mice, hamsters, rabbits and dogs with a wide variety of alpha and beta emitters. These studies have been reviewed in ICRP Publication 31 (1980) with some additional considerations in ICRP Publication 48 (1986). Some general conclusions from these studies are cited below. In the case of inhaled insoluble alpha-emitters such as plutonium, direct effects in the lungs include pulmonary fibrosis and lung cancer. Deposition in the lymph nodes and lymphatics leads to fibrosis of lymph nodes with loss of viable lymphocytes. The biological effects of inhaled soluble alpha emitters include bone cancer (ICRP 1980).

Lung fibrosis and respiratory insufficiency are major causes of premature death at high doses of inhaled insoluble alpha emitters; lung cancer is the only major cause of premature death in animals at low doses. The lowest dose levels at which statistically significant increases of lung cancer have been observed in experimental animals with inhaled insoluble alpha emitters are about 37 Bq per g lung (ICRP 1980).

Linear and nonlinear models used to evaluate the animal data would project to risk coefficients between 20 and 1600 lung cancers per 10^4 animals per Gy of alpha radiation (ICRP 1980). These values are generally supportive of the ICRP (1977) risk estimate of about 20 lung cancers per 10^4 humans per Gy of X- or gamma-rays or 400 lung cancers per 10^4 humans per Gy of alpha radiation. (The corresponding values from the more recent 1988 UNSCEAR and 1990 BEIR reports cover a range from 6 to 85 lung cancers per 10^4 person-Gy of low LET radiation at low dose rate, as compared to the 1977 ICRP value of 20.)

Comparison of the data from animal experiments in which lung cancer occurred following deposition of beta-gamma emitters and of alpha-emitters in the lung gave an equal effectiveness ratio of 30 for inhaled alpha-emitting radionuclides (ICRP 1980). Other comparisons of alpha and beta-gamma dose-effect models gave ratios of 6 to 40 (ICRP 1980). Thus the experimental animal animal data

on lung cancers following deposition of radionuclides in the lung tend to support the ICRP (1977) decision to change the recommended quality factor for alpha-particles from 10 to 20.

5. COMPARISON OF OCCUPATIONAL RISKS IN URANIUM MINING

5.1 Current Radiological Exposures of Uranium Miners

Data on recorded exposures at uranium mines in Canada were supplied by the National Dose Registry and are summarized in Table 7. These recorded exposures are not corrected either for potential errors in exposure measurements (this topic will be considered later) or for variations in the time that individual workers were employed in the mines in any given year. The exposures recorded in the National Dose Registry are those from external gamma rays as measured by personal thermoluminescence dosimeters, and from short-lived ^{222}Rn progeny, as estimated by grab samples for area monitoring. For purposes of a first tentative comparison with recommended annual limits, it might be assumed that the sum of the gamma exposures in cSv and of the radon progeny exposures in WLM should be less than 5. (The biological hazard from 1 cSv or 10 mSv of external gamma was previously assumed by the ICRP to be roughly equivalent to that from 1 WLM of radon progeny). The data in Table 7 suggest that average exposures in two underground uranium mines in Ontario are about one-third of this assumed limit, while average exposures in two open pit uranium mines in Saskatchewan are less than 5% of this assumed limit. In the Ontario underground mines, 1% of the workers may exceed 80-90% of this assumed initial limit (Table 7) while 99% of the workers in the Saskatchewan open pit mines would not exceed about 25% of this assumed initial limit.

As noted in the introduction, the ICRP has, however, recommended that annual limits should be based on the sum of exposures to all sources of radiation. The sum of the annual effective dose equivalents from external radiation and of the annual committed effective dose equivalent from internal radionuclides should not exceed 50 mSv (5 cSv).

This problem has been explored at one of the underground uranium mining operations in Ontario by Duport (1988 a). The annual limitation of stochastic effects for that particular facility can be calculated from the formula

$$\frac{I_E}{50\ \text{mSv}} + \frac{I_{Rn}}{4.8\ \text{WLM}} + \frac{I_{Tn}}{14\ \text{WLM}} + \frac{I_{U,Th}}{2400\ \text{Bq}} \leq 1,$$

where I_E is annual exposure to external gamma, I_{Rn} is annual exposure to short-lived radon progeny, I_{Tn} is annual exposure to short-lived thoron progeny, and $I_{U,Th}$ is annual intake of long-lived alpha emitters of the mixture of uranium and thorium ore dusts found at that facility. The annual limits for the first three terms in this formula are those recommended by the ICRP. The ICRP has recommended that annual limits on intake (ALI) of long-lived alpha activity in ore dusts for the limitation of stochastic effects should be 1700 Bq for uranium ore dusts and 380 Bq for thorium ore dusts with an activity median aerodynamic diameter (AMAD) of 1 μm. If there were approximately equal amounts of thorium and uranium in the ore, as does occur in certain Ontario mines, the ICRP recommendations would be closer to 1000 Bq (AMAD of 1 μm) than to 2400 Bq (AMAD of 5 μm) for the annual limit on intake of this particular ore dust. The ICRP

recommendations on ALI for long-lived ore dusts have stimulated considerable scientific research and the ALI values are currently believed to be too low for uranium mines in Canada; this topic was considered in more detail in section 3. In the state of our current knowledge, it seems that an ALI value of 2400 Bq for Elliot Lake ore dust might be more appropriate (Duport 1988b). This is probably a conservative estimate; the data discussed in section 3 suggest that the best value of the ALI for the typical ore dusts encountered in Elliot Lake Mines might be even higher, i.e., the risks from inhaled ore dust may be somewhat lower than those calculated using a stochastic ALI of 2400 Bq.

Using the above formula, it was estimated that 50% of the miners in one particular underground uranium mine at Elliot Lake would not be exposed to more than about 0.4 times the annual limit on the sum of the combined exposures (Table 8). The fraction of the workers in this particular mine who might be exposed to more than the annual limit as calculated by the above formula would be about 3% (Duport et al., 1988a). This conclusion must be taken with caution since it is based on the a posteriori examination of an exposure distribution curve and does not take into account actions that might have been taken by mine management in their routine review of individual exposures. An increase in the best ALI value for long-lived alpha emitters in ore dust would have little effect on these percentages, since the exposure to ore dust represents a small fraction of the combined exposures to all radiation sources in this particular underground uranium mine (Table 8).

A related analysis of 1982-83 data for two underground and two open pit mines in Canada was presented by Ching et al. (1985). Data on individual exposures to thoron progeny and to long-lived alpha emitters in ore dust were not available at that time. Assuming that 20 percent of the effective dose equivalent was contributed by the sum of these two latter sources, the number of workers exceeding the limit recommended by the ICRP would have been 18-32% of the total workforce in the two underground mines and 0% in the two open pit mines in 1982-83 (Ching 1985). Re-analysis of the situation using more recent data on exposures in underground mines in Ontario indicates that a very low percentage of workers would exceed the recommended limits in recent years (Ching 1989).

Further information on data derived from measurements of long-lived alpha-emitters in ore dust in two uranium mines in Canada in 1982-83 is given in Table 9. Obviously the inhalation of ore dusts can contribute appreciably to the total radiation exposure of underground uranium miners in Ontario. The situation for open-pit miners in Saskatchewan is not yet clear. Relevant data are summarized in Table 10. It should be noted that the relative thorium content of the high grade uranium ores in Saskatchewan is very low, in contrast to the low grade uranium ores mined at Elliot Lake in Ontario. Thus the contributions from thoron progeny do not present a major problem in radiological protection in the Saskatchewan mines, as they do in the Ontario mines (Table 8). In Saskatchewan mines, the contribution of thorium to the total dose, from inhalation of ore dust, arises from thorium-234, a decay product of uranium 238.

The data in Table 7 are based upon recorded exposure measurements. These recorded values are of course subject to many uncertainties. The measurements of external gamma exposures do not represent absorbed gamma doses in the tissues of the body. The correct values for absorbed dose equivalents of gamma radiation are probably about 0.6 to 0.8 times the recorded exposures (Jones, 1964;

Table 9 in ICRP 51, 1987) and would be even lower if the assumed quality factor for gamma-rays were less than 1 (ICRU 1986). Exposures to short-lived radon progeny are estimated from the concentrations of radon progeny found in grab samples of the air in different areas of the uranium mining and milling operation, and from estimates of the time that each worker spent in these areas. Measurements of exposures in an underground mine in Ontario based on personal dosimeters (Duport 1988a) indicated that annual exposures for 50% of the workers are about 0.8 times the estimated and recorded exposures based on a grab-sampling technique (Table 11). The recorded exposures can, however, be appreciably less than or greater than the measured exposures of individual workers, particularly over shorter periods of time than one year (Table 11). Another problem to be considered is the fact that current methods of measuring radon progeny include a portion of the exposures to thoron progeny (Berthelot 1989).

The methods currently used in Canada to estimate exposure of uranium miners to radon progeny probably provide reasonable values for the average exposures of the miner population. However, the variation in magnitude and time course of the exposures of individual miners appears to be large. Personal dosimeters for inhaled ore dusts as well as for inhaled radon and thoron progeny would of course be invaluable for assessment of the exposures of individual miners in future.

5.2 Conventional Risks

Mining in general, and underground mining in particular, is a hazardous occupation due to a relatively high rate of fatal and non-fatal accidents. Uranium mining is no exception to this general rule.

The ICRP (1985) has summarized the average rates of occupational fatal accidents in various categories of industry in Canada for 1972-81. These numbers are based on data compiled by provincial workers' compensation boards and do not include the 20% of workers in Canada who do not contribute to workers' compensation programs. The rates of occupational fatal accidents range from about 0.1 in finance to about 20 per 10^4 workers per year in fishing and hunting. Mining ranks third from the top with an average fatality rate of 5 per 10^4 workers per year. Deaths due to occupational illness also appear to be high in mining (ICRP 1985).

Rates of occupational accidental deaths for various groups of miners in Ontario are summarized in Table 12. The average for all mine workers in Ontario (Table 12) is similar to that for all of Canada (ICRP 1985). However, the rate of fatal occupational accidents for underground miners is somewhat higher and in the region of 9×10^{-4} per worker per year for Ontario (Table 12). The rates for underground miners in Sweden and the USA in 1970-74 were slightly higher than for Ontario (Ham 1976). The rate of fatal occupational accidents in open-pit mines is appreciably lower (Table 12). Mining is also associated with a relatively high rate of non-fatal compensable injuries (Table 12).

Data for uranium mines in Ontario are summarized in Table 13. These values are averages for all workers at the mines; approximately 59% of these workers appear to be underground miners (Table 7). Assuming that the ratios of accident frequencies for underground uranium miners compared to shop and surface workers are similar to those reported for all Ontario mines (Table 12), then the average

accident frequencies given in Table 13 would be equivalent to about 4.5 fatal accidents and 455 lost-time accidents per 10^4 underground uranium miners in Ontario over the years 1984-88. These particular values will be used in subsequent calculations. Accident rates for open-pit miners have been estimated very approximately from the data in Table 12.

5.3 Comparison of Risks

There are major problems associated with the estimation of the relative importance of fatal accidents, non-fatal accidents and other occupational hazards associated with uranium mining. This topic has been discussed in considerable detail in ICRP Publication 45 (1985), which recommended time-loss detriment as a basis of comparison. Rough estimates of years of life expectancy lost or seriously impaired due to occupational hazards are given in Table 14. Comments on the methods used to derive the numbers given in Table 14 follow.

(a) Cigarette smoking: Data on years of life expectancy lost due to cigarette smoking are derived from Hammond (1967), Kelsey (1978), Cohen and Lee (1979) and similar sources; the total loss is usually estimated at about 6 years per person after continuous smoking of one pack per day for 50 years. Although the data are based on analyses of the past experience of males in the general population, similar values would be expected for females with the same rate and duration of cigarette smoking. The years of life lost due to continued cigarette smoking are directly proportional to the number of cigarettes smoked per day. For comparative purposes, the data in Table 14 are given per year of continued exposure; it should, however, be noted that the additional health risk due to cigarette smoking would largely disappear within 20 years after cessation of smoking (Doll 1978).

(b) Fatal accidents: The detailed analysis in ICRP Publication 45 (1985) indicates an average of 35 years of life expectancy lost per fatal occupational accident. An average fatality rate of, for example, 1×10^{-4} per worker per year would thus result in an average of 0.35 years of life lost per 100 workers per year of work.

(c) Non-fatal accidents: The average time off-work for compensable non-fatal accidents is about 25 days (ICRP 1985, NSC 1988). There are significant problems with the interpretation of data on rates of non-fatal accidents. In the first place, the reported rates vary considerably from one jurisdiction to another and the reported rates for non-fatal occupational accidents in Canada appear to be increasing by about 2% per year while the rates for fatal occupational accidents are decreasing by about 4-5% per year (Myers 1985); the reported rates for non-fatal accidents are thus highly dependent on local interpretations of local compensation regulations. Reported rates of disabling injuries, as well as of fatal accidents, have decreased by about 2-fold since 1945 for workers off-the-job in the USA (NSC 1988). Secondly, and more importantly, non-fatal lost-time injuries can be subdivided into a majority of cases of temporary disability and a minority (about 3%) of cases of permanent total or partial disability. The small proportion of cases of permanent disability contribute about as much to total time loss as do the majority of cases of temporary disability (ICRP 1985, NSC 1988). For compilation of the relative detriments in Table 14, it was assumed that non-fatal lost-time

accidents would on average result in 50 days of serious impairment of life. Data on accident rates and time-loss detriment in safe industries were taken directly from ICRP (1985).

(d) Silicosis: Uranium ores in the Elliot Lake region in Ontario contain 60-70% quartz in the total dust, a concentration which is much higher than that in other hard-rock mines (Ham 1976). This has resulted in an appreciable incidence of silicosis among early uranium miners in Ontario. The time-loss detriment that might be attributed to silicosis is difficult to estimate. The number of deaths attributed to silicosis among the early Ontario uranium miners is about one-sixth of that attributable to radon progeny (Muller 1988) and the average age at death in miners with silicosis is similar to that of the average male in Canada (Myers 1985). As a very rough estimate, we have assumed that the years of life impaired due to silicosis under current conditions in the Elliot Lake mines might be about one sixth the years of life lost as a result of exposure to radon progeny (Table 14).

(e) Radiation-induced cancers: Estimates of annual exposures to radon progeny and external gamma radiation were taken from data recorded in section 5.1. Lifetime risk coefficients for radon progeny were taken to be about 3×10^{-4} per WLM (section 2.7) and for exposures to external gamma-rays about 6×10^{-4} per cSv (section 3.1). Estimates of additional radiation hazards from other sources, notably thoron progeny in air and long-lived alpha-emitters in ore dust, are very approximate and are based on the considerations discussed in sections 3 and 5.1. To calculate the time-loss detriment (Table 14), ICRP (1985) indicates 15 years of life lost plus 1 year of serious impairment per radiation-induced fatal cancer in workers; UNSCEAR (1988) indicates 14 years and 22 years of life lost per radiation-induced fatal cancer (including leukemia) in workers based on relative and absolute risk models respectively. The total loss of life expectancy per Sv due to radiation-induced fatal cancers is essentially identical for calculations based on relative and absolute risk models (UNSCEAR 1988). For the compilation of relative detriments in Table 14 it was assumed that each radiation-induced fatal cancer in workers would result in an average of 18 years of life lost or seriously impaired. The statistical risks due to exposure to external gamma-rays include non-fatal cancers and genetic diseases in children as well as fatal cancers; we have assumed on the basis of considerations discussed in ICRP (1985) that an average of 18 years of life lost or seriously impaired could be applied to the total risk coefficient of 6×10^{-4} per cSv for external gamma radiation. Since most uranium miners have traditionally been male, no additional detriment attributable to gamma irradiation of pregnant females (ICRP 1985) has been considered in Table 14.

The following conclusions can be drawn from Table 14: (i) Uranium mining is a relatively hazardous occupation. For underground uranium miners in Ontario, the total detriment due to occupational hazards is about 4 times greater than that attributable to employment in a safe industry (ICRP 1977) with a fatal accident rate of 10^{-4} per worker per year. (ii) Some 60%-70% of the detriment attributable to employment as a uranium miner appears to be due to conventional occupational accidents. (iii) Cigarette smoking represents a major voluntary health hazard for uranium miners as well as for other smokers. Moreover, the risks of lung cancer induced by radon progeny and of silicosis from quartz dust are smaller for non-smokers than for smokers (Muller 1986).

Some indication of the relative importance of fatal occupational accidents can be derived from Fig. 4. For all industries combined, fatal occupational accidents represent about one-quarter of all fatal accidents to workers (Fig. 4). For an average of 10^{-4} fatal accidents per worker per year, fatal occupational accidents would represent about one-thirtieth of deaths from all causes between ages 20 and 65 (Statistics Canada 1988). However, fatal occupational accidents in underground mining are higher than 1×10^{-4} per year and add a more significant proportion to the total deaths expected for persons of the same age in the general population.

6. DISCUSSION

The assessment of the risk resulting from occupational exposure to the short-lived progeny of radon-222 is affected by various confounding factors. The estimation of the risk per unit exposure is also affected by the choice of the exposure-response model used in the calculation and by errors in both the exposure data and the ascertainment of health effects. An alternative approach is to evaluate the risk in relation to dose to the exposed tissues rather than to exposure data. However, this approach depends, among other things, on data derived from epidemiology. It can therefore not be considered a purely "dosimetric" approach. Other problems with the "dosimetric approach" are uncertainties in physiological parameters used in the calculations. However, the dosimetric approach is the only feasible technique for deriving exposure-response functions for alpha emitters for which there are no satisfactory human observations, such as for short-lived progeny of thoron and for long-lived alpha emitters in ore dust.

The observation of the effect of exposure of human populations appears to be the most straightforward and simplest approach to study exposure-response relationships. There are, however, several serious pitfalls even in the use of the results of observations of human populations, the epidemiological approach. Observation extends over a limited period of time only, which is less than the average lifespan of the exposed group. Most of the individuals occupationally exposed to radon daughters are still alive, and the full extent of health effects has not been observed in most of these populations. There are also many errors and uncertainties in the historical exposure data used in these studies. Exposure is not limited to radon and its progeny only, but includes also other carcinogenic or possibly co-carcinogenic substances. Cigarette smoke, silica, and blasting and diesel fumes are examples of such substances. As a result, epidemiology is not fully adequate for deriving exposure-response functions unless particular care can be taken to account for each of these potential pitfalls.

The limited period of observation of occupational groups makes it necessary to extrapolate well beyond the period of actual follow-up. Various models can be used to do so and the results in terms of lifetime risk estimates will vary according to the model used. It is desirable to study the effects of attained age of the population, time elapsed since each individual increment in exposure, smoking habits, and exposure to other cancer causing agents. As many of these factors as possible should be taken into account in the epidemiological study. In order to do so, the appropriate raw data have to be collected and adequate models for the exposure-response relationships under various conditions derived. This obviously goes beyond simple observation and errors of judgment or interpretation are likely to occur.

A simple, linear, non-threshold exposure-response function is most frequently used in practice in radiation protection. The great advantage of this model is its simplicity and ease of application. In general, the epidemiological data on excess lung cancers induced by exposure of miners to radon progeny are compatible with this simple model, at least up to cumulative exposures of about 300 WLM.

Risk can be expressed as absolute or attributable risk per unit exposure or as incremental relative risk. The use of the absolute risk model implies that the incremental risk is proportional to exposure, but independent of the underlying "normal" risk. The relative risk model assumes that the incremental risk is proportional to both exposure and the underlying normal risk. The choice of the model is important for extrapolating beyond the period of observation. As indicated earlier, the risk factors derived from various epidemiological studies are not identical. A relative risk of 1% per WLM and an attributable risk of 10^{-5} per WLM per year are representative of the results obtained. Recent analyses have concentrated on a model in which the relative risk varies with time since exposure. However, review of the available data does not suggest any reason to change the previous lifetime risk estimates of 1-6 x 10^{-4} lung cancers per WLM (ACRP 1982). The best central value for inhaled radon progeny is probably close to 2.5-3 x 10^{-4} lung cancers per WLM for the average uranium miner in the age range of 20 to 60 (see section 2).

The biological effects of external gamma radiation are currently being discussed by a number of scientific committees and were not reviewed in detail in this document. Available data suggest that the serious biological consequences of exposure to 10 mSv (1 cSv) gamma rays are somewhat higher than those of exposure to 1 WLM radon progeny. Based on data available to the beginning of 1990, a reasonably conservative estimate of the potential risks of serious biological consequences of exposure of workers to gamma radiation at low dose rate would be equivalent to about 6 x 10^{-2} per person-Sv. The range of uncertainty in this value is from less than 1 x 10^{-2} to about 10 x 10^{-2} per person-Sv (see section 3.1).

Dosimetric calculations indicate that the stochastic risks per WLM of inhaled thoron progeny are about one-third of those for radon progeny. In other words, the best central risk estimate for inhaled thoron progeny is probably about 1 x 10^{-4} fatal cancers per WLM, with a range of uncertainty from about 0.3 to 2 x 10^{-4} per WLM (see sections 3.2 to 3.6).

Calculation of the relative risks of inhaled radioactivity in ore dusts is fraught with many uncertainties; in the present document, it was assumed that the stochastic risks associated with the inhalation of 2400 Bq of long-lived alpha-emitters in ore dust containing equal proportions of uranium and thorium would be roughly equivalent to a committed effective dose equivalent of 50 mSv. This value appears to be a reasonably conservative estimate for most ore dusts. The calculated values depend greatly on the size, composition and solubility of inhaled ore dusts in various uranium mines, as well as on the physiological parameters chosen for metabolic modeling of radionuclides in the body. Best values for the annual limit on intake of inhaled ore dusts in different uranium mines in Canada could vary from about 1000 to 30 000 Bq, depending on the parameters noted above (see sections 3.2 to 3.6).

Comparison of the relative impact of conventional accidents with those of radiation-induced cancers has been based on years of life expectancy lost or seriously impaired. The results of this comparison confirm previous suggestions that uranium mining is a relatively hazardous occupation. The total time-loss detriment due to occupational hazards in underground uranium mining is about 4 times greater, and in open-pit uranium mining slightly greater, than that attributable to employment in a safe industry with a fatal accident rate of 10^{-4} per worker per year. Conventional accidents probably account for about 60% of this total occupational detriment in underground uranium mines in Ontario in recent years.

The proposed amendments to regulations governing radiation exposures of uranium miners in Canada are based on an annual limit of 50 mSv total from all radiation sources (AECB 1986, 1989). These proposed regulations will be more restrictive, and thus potentially more protective of the health of uranium miners, than the past custom in some jurisdictions of applying separate, independent limits on exposures to external gamma radiation and to radon progeny.

The February 1990 draft of proposed new ICRP recommendations suggests that maximum occupational exposures should be reduced from 50 mSv per year to 100 mSv per 5 years (i.e., an average of 20 mSv per year). This change would have little effect on maximum occupational exposures to radon progeny alone. Assuming that the average risk from radon progeny is 2.5-3 x 10^{-4} WLM^{-1} and that the average weighted risk to workers from external gamma rays is 6 x 10^{-2} Sv^{-1}, then 20 mSv per year would be equivalent to 4-4.8 WLM radon progeny per year. The potential risks from other radiological hazards in uranium mines would have to be recalculated, using new weighting factors and new risk coefficients from ICRP. Preliminary assessment based on the February 1990 ICRP draft suggests that any changes in risks from thoron progeny and inhaled ore dusts would be relatively small.

Continued efforts to further reduce the radiation exposures of uranium miners are highly recommended. Total occupational hazards to underground uranium miners will, however, never be reduced to those characteristic of safe industries unless the frequency of traumatic accidents can also be further reduced.

Other risks such as those resulting from exposure to silica, noise and vibration should be taken into account when evaluating the total occupational risk in uranium mining. The objectives in considering all risks are to reduce the total risk from all causes and to use funds spent for health protection as effectively as possible, as suggested in paragraph 12 of ICRP Publication 55 (1989).

7. RECOMMENDATIONS FOR FUTURE WORK

(a) Further study of the relative impact of conventional accidents and other non-radiological hazards in uranium mining is required. A more efficient system should also be put in place to periodically update statistics on occupational accidents and illnesses, and to publish trend analysis in health and safety.

(b) Continued epidemiological follow-up of miners exposed to high concentrations of radon progeny in the past is required, particularly to assist in decisions on the most appropriate risk projection models for the estimation of lifetime risk per unit of exposure.

(c) Better definition of the hazards from ore dust is required. This would include assessment of the solubility, sizes and respirable fraction of ore dust particles, with particular attention both to silica content and content of long-lived alpha-emitters. Site specific ALIs for ore dusts in different uranium mines should be considered.

(d) An expert group should be established to recommend practical methods to reduce the total risk from all causes in uranium mining and to recommend how funds should be spent on the protection of health as effectively as possible.

(e) Because of the large uncertainties associated with estimates of individual exposures, the accuracy and reliability of exposure assessment methods should be investigated, and new, more reliable methods for determining individual exposures should be promoted.

(f) Radiological hazards from inhaled thoron progeny and from inhaled ore dusts should be recalculated when new weighting factors and new risk coefficients become available from ICRP.

Table 1

Estimates of risk of induced lung cancer in various groups of miners

Group (and approximate number) of miners	Average estimated exposure in WLM	Excess lung cancers appearing more than 5-10 years after first exposure: % increase in normal incidence per WLM	Excess lung cancers appearing more than 5-10 years after first exposure: Excess cancers per 10^6 person-years per WLM	Reference
Eldorado Port Radium uranium (2,103)	273[a]	0.27	3.1	(Howe 1987)
U.S. uranium (3,366)	1180	0.3-0.45 0.8-1.4[b]	3.5 6[b]	(BEIR 1980,1988, Whittemore 1983, Hornung 1987)
Nfld. fluorospar (1,772)	548	0.9	6.4	(Morrison 1988)
Ontario uranium (15,984)	33	1.0 1.3[c]	9.7 7.2[c]	(Muller 1985)
Czech uranium, group A, started 1948-52 (2,194)	303	1.8	19.0	(BEIR 1980)
Czech uranium, group S, (includes group A), started 1948-57 (4,043)	226	about 1.6	21.0	(Sevc 1988)
Malmberget iron (1,415)	94	1.4	20.0	(BEIR 1988, Radford 1984)
Eldorado Beaverlodge uranium (8,487)	36[d]	2.6-3.3	21.0	(Howe 1986, BEIR 1988)

a 44% of the person-years at risk in the category with <5 WLM and with no excess lung cancers were excluded for this calculation.

b Calculated for groups receiving lower exposures only.

c Risk coefficients for the group of miners with no prior experience in gold mining. Data based on "special WLM" estimates (Muller 1983,85) are not included.

d 74% of the person-years at risk in the category with <5 WLM and with no excess lung cancers were excluded for this calculation.

Table 2

Risk coefficients[a] for Czech uranium miners (group S) of different ages (Sevc 1988)

Age at start of exposure (years)	Age at death (years)	Total number of lung cancers	Relative risk coefficient	Absolute risk coefficient
up to 30	40-49	49	4.3	6.0
30-39	50-59	59	2.0	13.1
40 and above	60-69	79	1.3	25.2
all ages	all ages	341	1.5	20.0

[a] These risk coefficients are not corrected for a 5 year minimum latency period. Group 5 consists of 4,043 Czech uranium miners who started mining between 1948 and 1957.

Table 3

BEIR-IV data on excess lung cancers in four groups of miners at various times after last exposure in the mines

Group of miners		Time since last exposure		
		<5 years	5-15 years	>15 years
Beaverlodge	O/E	21/2.84	18/7.37	15/9.08
	(E.R.R.)	(6.4%)	(1.4%)	(0.65%)
Ontario	O/E	14/5.12	35/18.39	28/27.94
	(E.R.R.)	(1.7%)	(0.9%)	(0.0%)
Malmberget	O/E	25/4.28	15/4.94	11/5.54
	(E.R.R.)	(4.8%)	(2.0%)	(1.0%)
U.S.	O/E	70/11.32	132/24.37	54/23.43
	(E.R.R.)	(5.2%)	(4.4%)	(1.3%)

Table 4

Lifetime risk estimates for Canadian miners, adjusted to the risk coefficients given in ICRP Publication 50[a]

Age at exposure	Excess risk of death from lung cancer by age 70 per 10^4 WLM		Reference
	Relative risk model	Absolute risk model	
Age 30-35	2.9 [9.6]	2.5 [5.1]	(Howe 1986)
Age 20-50	2.8 [2.4]	2.6 [1.7]	(Morrison 1988)

a The original lifetime risk estimates are given in brackets in the table; these values were adjusted to the average risk coefficients of 1.0% per WLM (relative risk model) or 10 per 10^6 persons-years per WLM (absolute risk model) suggested in ICRP Publication 50 (1987).

Table 5

Comparison Between Past and Present Exposure Conditions[a]

	Pre 1958	Present
Radon daughters	0.3 to 1.4 WL	about 0.05 to 0.3 WL
Mineral dust	about 1 to 9 mg/m^3	0.05 to 1 mg/m^3

a Data derived from DMSA 1985 and Duport 1985

Table 6

Annual limit on intake recommended by the ICRP (1981,86) for various inhaled radionuclides

Radiation source	Annual limit on intake
Radon progeny	0.02 J of potential alpha energy (4.8 WLM)
Thoron progeny	0.06 J of potential alpha energy (14 WLM)
Uranium ore dust (1 μm AMAD)	1700 Bq of long-lived alpha activity
Thorium ore dust (1 μm AMAD)	380 Bq of long-lived alpha activity

Table 7

Recorded annual exposures at all underground uranium mines in Ontario and two open-pit mines in Saskatchewan

Date	Category of Workers	Number of Workers with Rn Exposure Records	Recorded Exposures* Mean (cSv)	Mean Rn (WLM)	99% Percentile (cSv)	99% Percentile Rn (WLM)
1986	Underground	1789	0.41	1.27	1.2	3.3
1985		1750	0.44	1.03	1.0	2.8
1986	All	3084	0.35	1.02	1.1	3.0
1985		2903	0.36	0.93	1.0	2.8
1987	Open-pit	334	0.04	0.06	0.2	0.21
1986		310	0.07	0.09	0.3	0.24
1985		279	0.06	0.06	0.3	0.25
1987	All	918	0.07	0.12	0.5	0.6
1986		838	0.07	0.17	0.4	0.8
1985		739	0.08	0.16	0.5	0.9

* Values for mean exposures were derived from data supplied by W.N. Sont, Risk Assessment Unit, National Dose Registry, Bureau of Radiation and Medical Devices, HWC. Data on exposures that would include 99% of the workers were derived from cumulative frequency curves plotted by P.J. Duport, AECB.

Table 8

Fractions of annual limits on exposure and dose received by 50 percent of the workers at one particular underground mine in Ontario*

Source	Fraction of Limit
External gamma	0.07
Radon (^{222}Rn) progeny	0.2
Thoron (^{220}Rn) progeny	0.04**
Long-lived alpha-emitters in dust	0.06
Sum	0.4

* Data derived from Duport et al. (1988 a). The values used to calculate annual limits are noted in the text.

** A recent report from another underground mine in Ontario indicates that over 50% of the workforce receives a thoron daughter exposure greater than 0.1 of the annual limit, but that the likelihood of thoron daughter exposure exceeding 0.3 of the annual limit is very small (Berthelot 1989).

Table 9

Average exposures to long-lived alpha-emitters in ore dust at two underground uranium mines in Ontario over a 12 month period in 1982-83*.

Mining Operations	Exposures as fraction of an ALI of 2400 Bq	
	Mean	Maximum
Underground 1	0.06	0.5
Underground 2	0.04	0.15

* Data from personal communication (Duport 1989).

Table 10

Mean intake of long-lived alpha-emitters in ore dust at different facilities, expressed as a fraction of ALI of 2400 Bq (data from personal dosimetry)*

Facility	mean intake	5% - 95% range of intake
Ontario underground A-1	0.01	0.003 - 0.04
Ontario underground A-2	0.08	0.003 - 0.2
Saskatchewan open-pit B	0.05	0.003 - 0.18
Saskatchewan open-pit C	0.01	0.003 - 0.04

* Data from personal communication (Duport 1989).

Table 11

Ratio of exposures to radon progeny (in WLM) as measured by personal dosimetry divided by those measured by grab sampling at an underground uranium mine in Ontario, for quarterly and annual exposure data*

Percentile	Ratio	
	Quarterly	Annual
10	0.1	0.4
50	0.6	0.8
90	1.25	1.4

* Data derived from Duport et al. (1988 a).

Table 12

Comparison of accident rates for various groups of workers in Ontario
(data for 1970-74 from Ham 1976; data for 1984-88 from Shulman 1990).

Category	Accident Frequency per 10^4 workers or per 2×10^7 man-hours per year		
	Fatalities		Non-fatal compensable injuries
	1970-74	1984-88	1970-74 (a)
Manufacturing	0.66	0.4	-
Construction	3.0	2.2	-
Logging and milling	15.7	7.2	-
Mining:			
reduction plants	0.9	0.6	330
shops and surface	1.3	2.0	568
open-pit operations	3.2	0.0 (b)	824
underground operations	8.9	8.8	1338
average	4.3	4.4	898

(a) Comparable data are not available for 1984-88 (Shulman 1990).

(b) Fatal accident rates for open pit operations are not very reliable since they are based on 0.14×10^4 worker-years at risk over the 5-year period 1984-88, as compared to 3.1, 4.9 and 5.3×10^4 worker-years at risk in reduction plant, shops and surface, and underground operations respectively (Shulman 1990).

Table 13

Accident Rates for Uranium Mines in Ontario (MAPA 1989)

Years	Accident frequency per 10^4 workers per year (a)	
	Fatalities	Lost time injuries
1978-83	4.3	627
1984-88	2.9	348

(a) Assuming 2000 hours per workers per working year (Ham 1976).

Table 14

Approximate estimates of time-loss detriment for various groups of workers

Category of workers	Hazard	Assumed annual exposure	Years of life lost or seriously impaired per 100 workers per year of continued exposure
All male workers	Cigarette smoking	365 packs (1 pack/day)	12.
Underground uranium miner	Fatal accident	4.5×10^{-4}	1.6
	Non-fatal accident	0.05	0.6
	Radon progeny	1.2 WLM	0.65
	Gamma rays	4 mSv	0.43
	Other radiation	about 6 mSv	0.65
	Silicosis	-	0.1
			4.0
Open-pit uranium miner	Fatal accident	1.5×10^{-4}	0.5
	Non-fatal accident	0.05	0.6
	Radon progeny	0.07 WLM	0.04
	Gamma rays	0.6 mSv	0.06
	Other radiation	about 2 mSv	0.2
			1.4
Safe industry	Fatal accident	$\leq 1 \times 10^{-4}$	$\leq$ 0.35
	Non-fatal accident	- .	$\leq$ 0.8
			$\leq$ 1.1

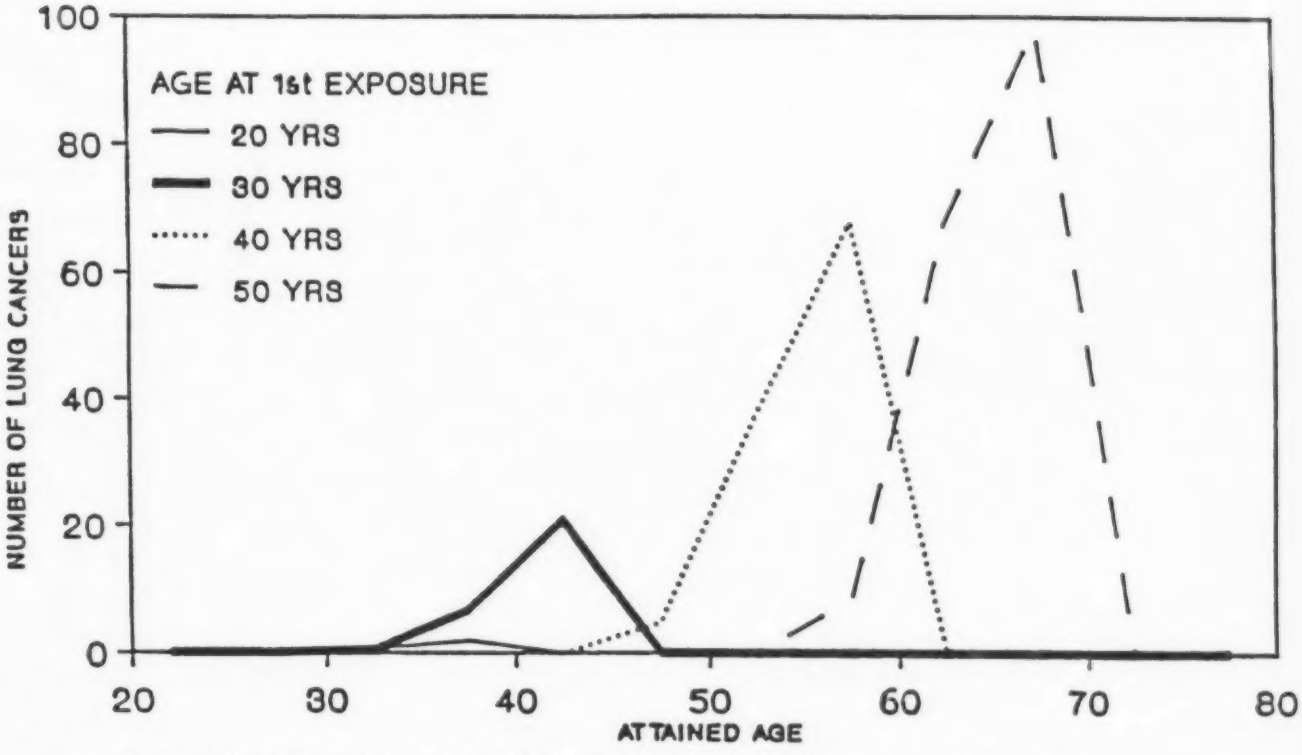

Fig. 1 Estimated risk of radiation-induced lung cancers in male uranium miners exposed to 1 WLM per year for 5 years starting at age 20, 30, 40 and 50 years (Muller 1989). Data are calculated per 100 000 males at birth from Canadian vital statistics, using relative risk estimates which vary with time since exposure as derived from the study of Ontario uranium miners (Muller 1989).

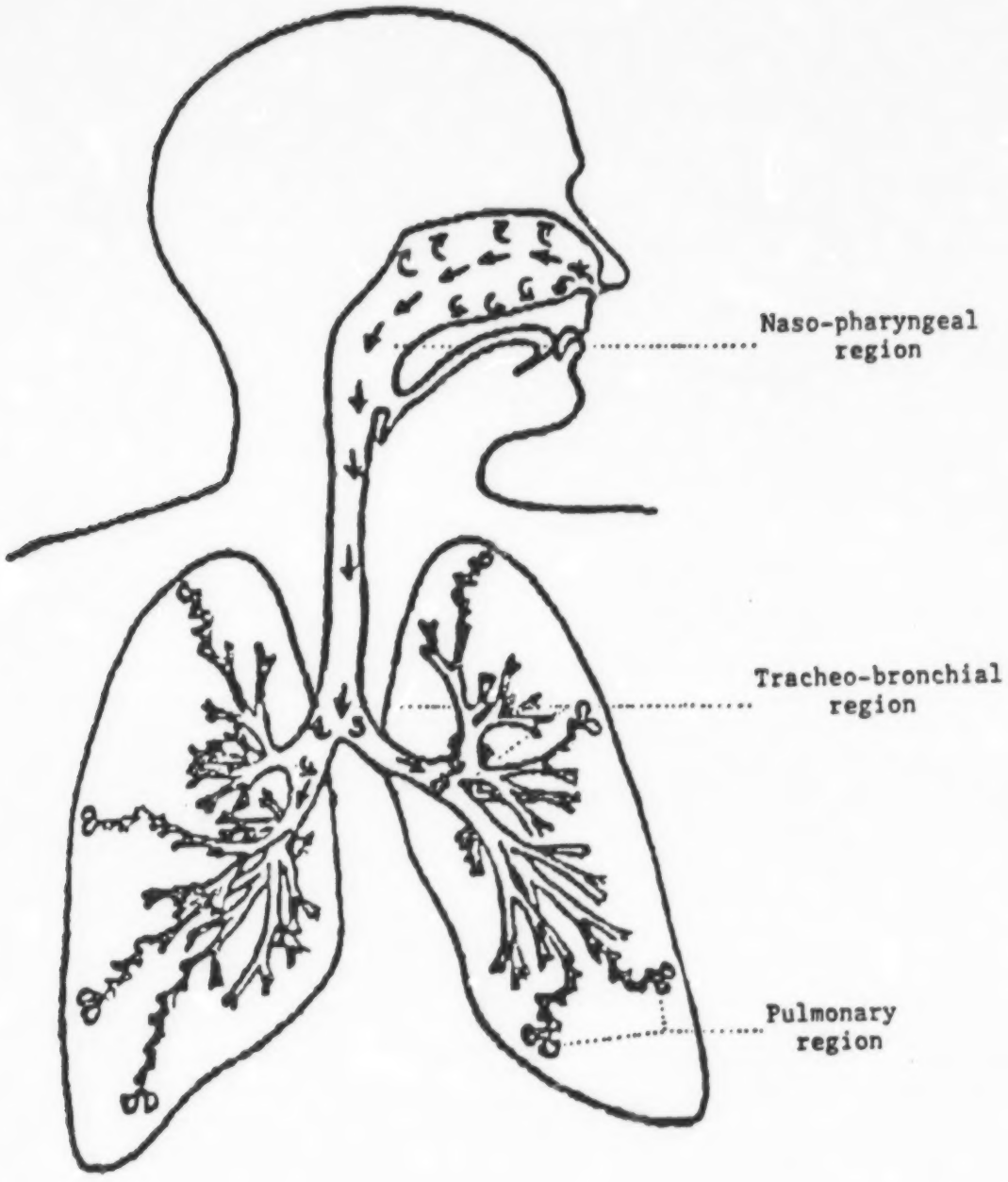

Fig. 2. Schematic diagram of the human respiratory tract.

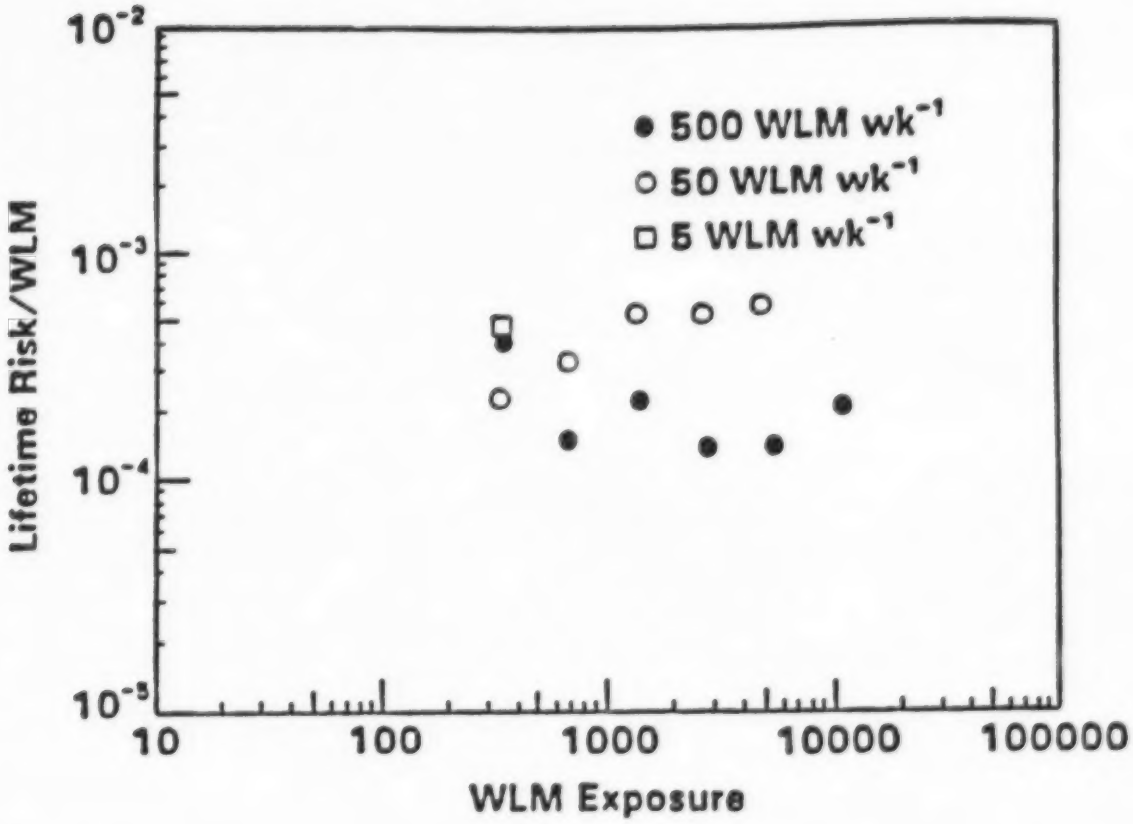

Fig. 3. Lifetime risk of lung tumors in male rats exposed to radon progeny, as derived from studies carried out at Battelle Pacific Northwest Laboratories (Cross 1988). Data adjusted for life-shortening.

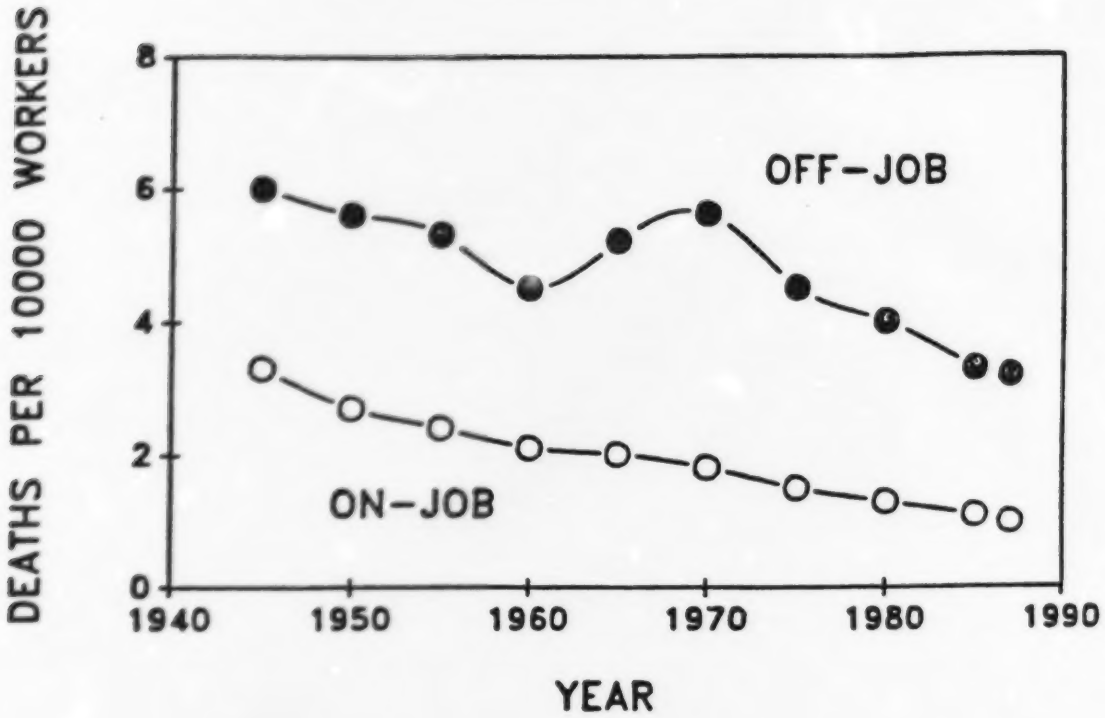

Fig. 4. Rates of fatal accidents for U.S. workers on the job and off the job over the years 1945-1987 (NSC 1988). The data are expressed in terms of total fatalities; rates per hour of exposure would be similar for workers on-the-job and off-the-job.

GLOSSARY

"absorbed dose": see "dose equivalent";

"activity": the rate of disintegration of a radioactive substance;

"Activity Median Aerodynamic Diameter" (AMAD): the value of the "aerodynamic diameter" that divides a distribution of particles such that half of the activity is carried by particles larger than the AMAD and half is carried by particles smaller than the AMAD.

"aerodynamic diameter": refers to a particle that behaves aerodynamically (settling velocity in air for example) like a spherical particle of unit density of the said diameter;

"Annual Limit on Intake" (ALI): the amount of radioactive substance taken into the body in one year that will result in a "committed dose" equal to the relevant annual dose limit;

"becquerel" (Bq): the special name of the unit of activity; one becquerel of a radioactive substance undergoes radioactive decay at the rate of one disintegration per second; (1 curie = 3.7×10^{10} Bq);

"class Y": a substance that is retained in the lungs for a period of years; more specifically, the time necessary to reduce the lung burden by half is longer than 500 days;

"committed dose": the total dose received from radioactive substances in the body during the 50 years following the intake of those substances;

"CEDE": committed effective dose equivalent;

"detriment": the mathematical expectation of harm incurred from exposure to radiation, taking into account the probability of each type of deleterious health effect and the severity of each effect;

"dose": a dose of radiation, either "dose equivalent", "committed dose equivalent", "effective dose equivalent", or "committed effective dose equivalent", depending on the context;

"dose equivalent": the "absorbed dose" multiplied by a "quality factor" to account for the different potential for injury of different types of radiation, the absorbed dose being the amount of energy absorbed in the body, an organ or a tissue due to exposure to ionizing radiation, divided by the respective mass of the body, organ or tissue;

"effective dose equivalent" (EDE): the sum of the "weighted dose equivalents" received by the organs and tissues of the body, the weighted dose equivalent being the dose equivalent to an organ or tissue multiplied by a weighting factor (W_T) in order to account for the risk of fatal cancer or inheritable injury resulting from irradiation of that organ or tissue compared to the total risk of such effects resulting from the receipt of an equal dose equivalent by the whole body;

"non-stochastic effects": biological effects which occur only if a certain threshold dose is exceeded, and for which the severity increases with increasing absorbed dose; e.g., reddening of the skin, vascular damage to tissues, opacities of the eye lenses, and acute radiation sickness;

"non-stochastic limit": a limit on dose equivalent that is intended to prevent the occurrence of non-stochastic effects;

"potential alpha energy": the energy of all the alpha particles that could be released from a given quantity of radon or thoron daughters during their successive stages of radioactive decay to lead-210 and lead-208, respectively;

"quality factor": see "dose equivalent";

"radiation": "ionizing radiation", i.e., any atomic or subatomic particle or electromagnetic wave having sufficient energy per particle or photon to produce ions in the material in which it is absorbed; ionizing radiation includes alpha and beta particles and gamma radiation, as well as neutrons and some other particles;

"radon" (^{222}Rn): the radioactive gas produced by the radioactive decay of radium-226 (which has been produced through a series of radioactive decay steps from uranium-238) and which, through radioactive decay, gives rise to "radon daughters";

"radon daughters": the following short-lived radioactive substances produced by a series of radioactive disintegrations from radon-222: polonium-218 (radium A), lead-214 (radium B), bismuth-214 (radium C), and polonium-214 (radium C');

"radon progeny": radon daughters;

"sievert" (Sv): the special name of the unit of "dose equivalent" and of "effective dose equivalent" (1 Sv = 1000 millisieverts = 100 rems);

"stochastic effects": biological effects, primarily cancer and genetic (inheritable) effects, that may occur at random amongst an exposed group of people, with the probability of occurrence usually being considered as proportional to the effective dose equivalent;

"stochastic limit": a limit on effective or weighted dose equivalent that is intended to limit the chance of occurrence of stochastic effects;

"thoron" (^{220}Rn): the radioactive gas produced by the radioactive decay of radium-224 (which has been produced through a series of radioactive decay steps from thorium-232) and which, through radioactive decay, gives rise to "thoron daughters";

"thoron daughters": the following short-lived radioactive substances produced by a series of radioactive disintegrations from radon-220: polonium-216 (thorium A), lead-121 (thorium B), bismuth-212 (thorium C) and polonium-212 (thorium C');

"thoron progeny": thoron daughters;

"weighted dose equivalent": see "effective dose equivalent";

"weighting factor": see "effective dose equivalent";

"working level" (WL): a unit of concentration of radon or thoron daughters in air expressed in terms of the "potential alpha energy" of the amount of daughters in a cubic meter or a litre of air; 1 WL = 2.08×10^{-5} joule per cubic meter = 1.3×10^{5} MeV per litre;

"working level month" (WLM): 170 working level hours (WLh), a WLh being a unit of exposure of a person to radon or thoron daughters; the number of WLh is determined by multiplying the concentration in working levels by the duration of exposure in hours.

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